be managed by giving supplemental oxygen. The major criterion for ventilatory support is the severity of acute respiratory acidosis. Generally if the pH falls below 7.25, mechanical ventilation is indicated. Hypercapnia with a compensated acidotic base state—pH 7.4—is not an indication for mechanical ventilation.

The major goals of ventilation are to provide adequate gas exchange while the precipitating cause of the respiratory failure can be managed. Many authorities now recommend that ventilatory support should also aim to rest the ventilatory muscles. The basis for this suggestion is that fatigue of the respiratory muscles is considered a complicating factor in the pathogenesis of respiratory failure. Recovery from the acute episode and facilitation of weaning may depend on providing adequate rest to the respiratory muscles. This thinking has influenced the modes of ventilation now in use.

Until recently, the major modes of ventilation available consisted of control, assist-control, and intermittent mandatory ventilation. The original promise of intermittent mandatory ventilation to improve patient ventilator synchrony and prevent the development of posthypercapnic metabolic alkalosis during ventilation has not been realized. Furthermore, it has not been shown to be superior in weaning patients from mechanical ventilation. Assist-control ventilation provides only partial rest to the respiratory muscles. If the respiratory drive is high, considerable work is expended by a patient.

Pressure support ventilation is a new mode of ventilation whereby a constant airway pressure is applied to the airway during spontaneous breathing or while using the intermittent mandatory ventilation mode. The patient is able to control tidal volume and inspiratory flow rates. The higher the level of pressure support applied, the larger the tidal volume and minute ventilation and the greater the reduction in patient energy expenditure. Low levels of pressure support—4 to 8 cm of water—are considered helpful in overcoming the resistance of artificial airways and ventilator circuits. When these levels of pressure support are used in intubated patients during spontaneously initiated breaths, it may be easier to evaluate the patient's ability to sustain spontaneous ventilation after extubation. Ongoing research will clarify the role of pressure support in improving patient ventilator synchrony, alleviating respiratory muscle fatigue, and facilitating weaning.

Once mechanical ventilation is started, the initial ventilator settings, rate, and tidal volume should be adjusted to maintain a normal pH. In cases of acute hypercapnia, carbon dioxide levels can be reduced rapidly to baseline to normalize the pH. In patients with chronic respiratory acidosis—a rapid reduction in the $Paco_2$ will cause an acute respiratory alkalosis superimposed on the present acid-base state and should be avoided. In this state, seizures and death can occur. Patients should be "reset" at their maintained $Paco_2$ before being weaned from the ventilator.

Higher inspiratory flow rates decrease inspiratory muscle work, reduce the physiologic dead space, improve arterial oxygen tension, and provide increased exhalation time and therefore a reduction in intrinsic positive end-expiratory pressure (PEEP). Intrinsic PEEP occurs with airway obstruction, delayed exhalation, and a consequent increase in the functional residual capacity. Successive breaths arrive prematurely before the previous expiration is complete, and intrathoracic pressure does not return to atmospheric levels, remaining persistently elevated, as when external PEEP is applied. Intrinsic PEEP is exceedingly common in patients with obstructive airways disease. Under normal circumstances, the pressure gauge on the ventilator records atmospheric pressure during expiration and reads zero even in the presence of high levels of intrinsic PEEP. The level of intrinsic PEEP can be measured by occluding the expiratory valve at the end of exhalation and noting the pressure on the ventilator gauge before the next inhalation. Intrinsic PEEP also causes distortion of the measurement of central venous pressure and lung compliance.

The mortality of chronic obstructive pulmonary disease in patients who require mechanical ventilation for acute respiratory failure ranges from 21% to 42%. About 3% of those who survive require long-term mechanical ventilation.

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Pulmonary Disorders Caused by Illicit Drug Use

ILLICIT DRUGS may be administered intravenously, intranasally, orally, or through inhalation by smoking. Pulmonary disorders associated with the intravenous and intranasal abuse of drugs have been well documented. Recent attention has focused on the effects of inhaling these drugs. There have been a number of recent reports of pulmonary disorders attributed to the inhalation of marijuana, cocaine, and methamphetamines.

Inhaling marijuana smoke may result in injury to the conducting airways as a result of noxious gases, oxidants, and other toxins in the particulate phase. Pathologic effects include ciliary dysfunction, inflammation of airway walls, squamous cell metaplasia, and peribronchial fibrosis. The large number of toxic substances produced during pyrolysis is similar to that produced during cigarette smoking. The physiologic effects of habitual abuse include increased airway resistance, ventilation-perfusion abnormalities, and decreased gas exchange surface, resulting in symptoms of cough, dyspnea, wheezing, and diminished exercise tolerance. The concentrations of some carcinogens and other toxins are greater in marijuana smoke than with tobacco, which may place regular marijuana users at a higher risk for bronchogenic carcinoma.

Cocaine hydrochloride must be converted to the pure alkaloid form ("crack") before it can be heated, volatilized, and inhaled. Abusers may have chronic cough, dyspnea, acute pulmonary hemorrhage, or, rarely, pulmonary edema. Patients may present with acute bronchospasm indistinguishable from an acute asthmatic attack from other causes. A decrease in the diffusing capacity of the lung for carbon monoxide (D_LCO) is the most commonly cited physiologic abnormality. The underlying mechanisms of decreased D_LCO from cocaine use are not well understood. Long-term cocaine smoking may cause a syndrome of fever, cough, and radiographic findings of diffuse pulmonary infiltrates. The pathophysiology of this clinical syndrome is postulated to be either from the direct action of cocaine or from volatile hydrocarbons. Dyspnea may also be a result of a pneumothorax caused by smoking cocaine and is thought to be due to the Valsalva's maneuver commonly practiced by these smokers.

Methamphetamine hydrochloride must first be highly purified to have the volatility necessary for inhalation. The purified form has a crystalline appearance and is commonly called "ice." This inhalation drug problem is epidemic in Hawaii and on the West Coast. The data on pulmonary complications, both acute and chronic, are limited. Physiologically, methamphetamine is a direct central nervous system stimulant that also increases sympathetic nervous system activity and serum catecholamine concentrations. Smoking "ice" results in central nervous system symptoms of euphoria, agitation, and psychosis and cardiovascular symptoms of hypertension, tachycardia, and arrhythmias. Although a direct effect of methamphetamine on lung tissue has not yet been proved, acute dyspnea and cough are associated. Because smoking "ice" is a relatively new phenomenon, it is possible that some yet-undescribed pulmonary or radiographic finding may occur.

In patients with unexplained cough, dyspnea, or radiographic findings, clinicians should consider illicit drug abuse. A urine immunoassay can detect cocaine or methamphetamine metabolites as long as two days after use.

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Mechanical Respiratory Support at Home

LONG-TERM HOME VENTILATOR SUPPORT is an attractive option for many patients with severe stable respiratory failure due to a variety of neuromuscular, musculoskeletal, and pulmonary diseases. Guidelines and standards now have established reasonable indications, selection criteria, protocols, and the team approach needed to prepare for home care and for follow-up.

When ventilator support is initiated electively, patients are able to adjust more easily, with assistance from trained family members. Elective ventilator support is usually needed less than 12 hours a day. Although tracheostomy has been used frequently, noninvasive methods should be considered first, such as positive pressure ventilation using a nasal mask or mouthpiece; negative pressure ventilation using a cuirass, iron lung, or various other techniques; and a rocking bed. Recent experience with the nasal mask suggests its advantages in effectiveness and in avoiding upper airways obstruction. The cost involved in ventilator assistance on an elective basis is within reach of usual resources for many patients, about $20 to $40 a day.

In contrast, patients in intensive care units on discharge require long-term ventilator support, usually 12 to 24 hours a day. A tracheostomy is often needed both for ventilation and to remove secretions. Patients may have multiple impairments and limited functional performance. Because more complex care is required, patients are more often dependent on others for most of the 24 hours. Paid caregivers are generally required to assist the family. The financial costs—about $150 to $300 a day—are greater than most patients can arrange, unless third-party or public funds are available, but are still less than the costs for hospital care. Adjusting to the unexpected and catastrophic situation is difficult for patients and families.

Hospital discharge preparation is a multidisciplinary team process that can enable most of these patients to go home or to another community alternative site. Experienced medical centers can assure a quality of care in the home environment that is comparable to that in a hospital. Two-year survival can be more than 70% for patients with neuromuscular disorders and more than 50% for patients with chronic obstructive pulmonary disease. Care must be coordinated with community agencies, a medical equipment company, and emergency services including the utility company, fire department, and a hospital emergency department. Patients generally prefer their quality of life at home when compared with the hospital setting; they can live in ways that are more personally satisfying and socially useful.

The challenges and burdens for a patient's family and the community home care resources can be enormous, particularly when care is complex and carried on for several years. The funding and community services required for these patients and their families need to be improved. Properly planned, however, long-term home respiratory support can be rewarding for patients, their families, and the medical team directing care.

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Inhaled Pentamidine Therapy for *Pneumocystis carinii* Pneumonia

INHALED PENTAMIDINE THERAPY is now indicated for the primary and secondary prophylaxis of *Pneumocystis carinii* pneumonia in patients with human immunodeficiency virus infection. Both primary prophylaxis, for persons with fewer than 2×10^6 per liter (200 per μl) CD4 (helper) lymphocytes who have not had an episode of *P carinii* pneumonia, and secondary prophylaxis, for those patients who have had a previous episode of the disease, are administered through a Respirgard II jet nebulizer (Marquest, Englewood, Colorado) at a dose of 300 mg of pentamidine isethionate once a month. This regimen is the result of a recent study of inhaled pentamidine as secondary prophylaxis for *P carinii* pneumonia that found giving 300 mg once a month superior to 30 mg or 150 mg given every two weeks. Relapse rates at 18 months were approximately 10% in patients receiving 300 mg once a month compared with a 65% relapse rate at one year in historical controls.

Toxicity from inhaled pentamidine has been minimal and limited to bronchospasm, preventable with the previous administration of inhaled bronchodilators.

Relapses of *P carinii* pneumonia after inhaled pentamidine therapy tend to occur in the apices of the lungs and possibly are related to poor drug deposition in those areas. Spontaneous pneumothorax (probably due to rupture of pneumatoceles in the apices) and extrapulmonary pneumocystosis have been reported in patients receiving inhaled pentamidine, raising a concern that pneumocystosis is in fact a systemic process and not one localized to the lung. Therefore, a combination of parenteral and local (inhaled) prophylaxis may be required. Two ongoing prophylaxis tri-